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Aging and Induced-Sputum Cells

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communications to the editor

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Detection of Hypoventilation

To the Editor:

The study by Fu et al (November 2004)[1] documents well that supplemental oxygen impairs the detection of hypoventilation by pulse oximetry, as discussed in the accompanying editorial by Demers.[2] We regularly perform overnight oximetry on inpatients to screen for sleep apnea/hypopnea and find that the results are very insensitive if performed using supplemental oxygen. Therefore, we perform overnight oximetry either using room air or, if the baseline awake oxygen saturation level is $< 90\%$, using only enough oxygen to bring the awake saturation to approximately 90%.

Fortunately, it is now possible to directly assess hypoventilation using continuous transcutaneous carbon dioxide tension monitoring.[3] We now routinely monitor transcutaneous carbon dioxide pressure in patients who are at high risk for hypoventilation in our ventilator weaning program. We find monitoring to be very helpful during the initial tracheostomy using a mask or during overnight periods when the patient is not receiving ventilation, as well as during bronchoscopies. The advantages of monitoring cutaneous carbon dioxide tension over monitoring with end-tidal carbon dioxide tension, which we also use, include allowing continuous measurement, not requiring deep exhalation, and making accurate measurements in patients with high dead space ventilation.

The device (CO-OXSYS Monitor, SenTec AG; Therwil, Switzerland) has been used in Europe[4,5] and is now available in the United States (Aspen Medical Products Inc; Irvine, CA). The monitor allows us to closely follow transcutaneous carbon dioxide pressure using a small probe that clips on the ear lobe. It usually takes about 5 min to equilibrate and then tracks carbon dioxide pressure closely, along with oxygen saturation.

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Aging and Induced-Sputum Cells

To the Editor:

We read with interest the article by Thomas et al[1] (December 2004) on the influence of age on induced sputum in normal subjects. We think that the discussion on the possible physiopathologic mechanisms could be deepened. Although an influence of advancing age on lung cellularity in healthy subjects has been already described,[2] reference values for cell counts in induced sputum in healthy adults > 50 years old are not available. The possible explanations of the results found by Thomas et al[1] could be an impairment in humoral lung immunity in older healthy subjects compared with younger healthy subjects,[3] and the presence of a low-grade inflammation in the lower respiratory tracts of many asymptomatic, older subjects.[4] In particular, a previous study[2] has reported in BAL fluid of the older healthy individuals an increase in CD4+/CD8+ lymphocytes ratio probably due to a repeated antigenic stimulation or irritation by environmental substances of the immune cells in the lower respiratory tract during the years. The recurrent antigen stimulation on the immune cells in the lung could be demonstrated by the decreasing with age of CD19+ B lymphocytes that represent the B cells not yet differentiated into antibody-secreting cells, suggesting that B cells on mucosal surface of airways in older subjects has been driven to differentiate by previous repeated antigen stimulations. The low-grade inflammation in the airways observed in older subjects might be related to the decline in the lung function that starts in the fourth to fifth decade of life in normal never-smoker humans. The mechanism by which neutrophils are recruited to within the airways in older healthy subjects is still unclear. A number of neutrophil chemoattractants can be secreted by inflammatory cells that reside in the airways, and epithelial cells can release cytokines, such as IL-8, which have a potent chemoattractant activity for neutrophils.[5] Low-grade persistent inflammation may occur because of the loss of factors that normally down-regulate the inflammatory response to pollutants

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or repetitive antigenic stimulations, combined with advancing age. Epithelial cells could be a significant source of neutrophil chemoattractants, which contributes to a low-grade inflammation in older subjects. Persistent, low-grade inflammation could damage elastin and perhaps lead to the age-associated loss of elastin fibers. Therefore, considering that many patients affected by asthma or COPD who increasingly perform induced sputum are often > 50 years old, these findings deserve further investigations.

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Ribavarin Should Be Tested in Clinical Trials in Combination With Other Antiviral Agents for Severe Acute Respiratory Syndrome

To the Editor:

We read with interest the article in *CHEST* by Chiou et al (July 2005)[1] and offer the following comments. The ribavirin-treated patients had higher lactate dehydrogenase levels, a well-known adverse prognostic factor in severe acute respiratory syndrome (SARS). The nonsignificantly higher mortality could be due to the more severe disease in this group. Viral load, another important predictor of mortality, was not available.[2] Moreover, Figure 1 seemed inaccurate: the survival in ribavirin-treated patients should be 0.88 at day 30 (5 of 44 patients died) instead of 0.71.

Classifying the ribavirin-treated patients into hypoxemic and nonhypoxemic subgroups (Table 2) and attributing the higher mortality in the hypoxemic subgroup to ribavirin was problematic, as both subgroups were treated with an identical protocol of ribavirin. From the data presented, a more likely explanation for the more severe drop in hemoglobin in the hypoxemic subgroup was that they had more severe disease. The survival curves in Figure 4 also appeared inaccurate: the survival in patients with drop in hemoglobin > 2 g/dL should be 0.69 (5 of 16 patients died) instead of 0.45. Hence, the result of the log-rank test ($p = 0.007$) needs to be justified.

Only factors that were potentially associated with hypoxemia were analyzed in Table 2. No univariate or multivariate analyses on factors related to death were reported. The conclusion that hemoglobin level was the only factor associated with death was not supported by the data presented.

In Figure 6, the shaded triangles were supposed to represent the hemoglobin of patients who were hypoxemic and had received ribavirin. There were 22 triangles, but there should only be 17 patients. In addition, expressing the survival of individual patients by proportion (y-axis) is difficult to understand.

Therefore, there is no convincing evidence that ribavirin has contributed to a life-threatening drop in hemoglobin or mortality in this report. As of today, three independent studies[3–5] have shown ribavirin to have *in vitro* activities against SARS-coronavirus, alone or in combination with other agents. Ribavirin should be tested in future randomized controlled studies in combination with other potential antiviral agents for SARS.

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A Modified Percutaneous Tracheostomy Technique Without Bronchoscopic Guidance

A Note of Concern

To the Editor:

We read with interest the article in *CHEST* by Paran and colleagues (September 2004)[1] on a modified percutaneous tra-

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